

NOT TO BE TAKEN AWAY

ST. MARK'S
LIBRARY
HOSPITAL

THE
BRITISH JOURNAL
OF

CHILDREN'S DISEASES.

VOL. XII.

MARCH, 1915.

No. 135.

Original Articles.

THE ATONIC FORM OF CEREBRAL DIPLEGIA.

By FREDERICK E. BATTEN, M.D.Cantab., F.R.C.P.,

*Physician to the National Hospital for the Paralysed and Epileptic, and to the
Hospital for Sick Children, Great Ormond Street; and*

WALTER H. VON WYSS, M.D.Basle, L.R.C.P., M.R.C.S.,

House Physician to the Hospital for Sick Children, Great Ormond Street.

FOERSTER described, in 1909, a new type of cerebral diplegia and gave detailed descriptions of four cases.

The main characteristic of this condition is an extensive hypotonia of the voluntary muscles of the body as shown by a large degree of undue passive mobility of the various joints. This hypotonia may be so extensive that even the muscles of the head and neck are affected.

There is complete inability to stand, walk, or even to sit without support. There is no power of fixation, owing to deficient action of the antagonistic muscles. When lying in bed the children are well able to move the limbs, though the movements are somewhat sudden. The tendon reflexes are present and the electrical reactions of the muscles normal.

All the children described by Foerster were mentally deficient. Two of them were completely mute at the age of three. One child of

two years was able to speak a few words, and the eldest child, aged 7 years, was able to speak, though with defective articulation. All of them were unclean in their habits. In one case there was hypotonia of the legs associated with a spastic condition of the arms and extensor response of the right plantar reflex. There were also occasionally opisthotonos and choreic movements of the face and tongue. One of Foerster's photographs shows a child in a peculiar position:



FIG. 1 (Case 1).—Shows the position of rigid extension of the legs produced when child is held up by axillæ.

he is held up by the axillæ, and the legs are extended at the knees and flexed at the hips. One child died at the age of three years, after an epileptic attack with a left-sided hemiplegia. One other died when two years old from measles. The pathological examination showed in both cases an extensive sclerosis of the frontal lobes without any affection of the cerebellum. No microscopical examination was made.

Pierce Clark, in 1913, described a similar condition under the name of cerebro-cerebellar diplegia. All his cases show the same

extensive hypotonia and inability to stand and walk. They were unclean in their habits, perfectly mute, and mentally deficient. All of them showed during the first few weeks of life inactivity of movements, whereas, later on, their power of movement was surprisingly good. If held up by the axillæ, the children showed a sort of epileptoid trepidation of the lower limbs. The author concludes on general anatomical and experimental evidence that the pathology of this condition is explained by a simultaneous lesion of both the cerebellum and the cerebrum.

Another case of this kind has been demonstrated by Thomas and

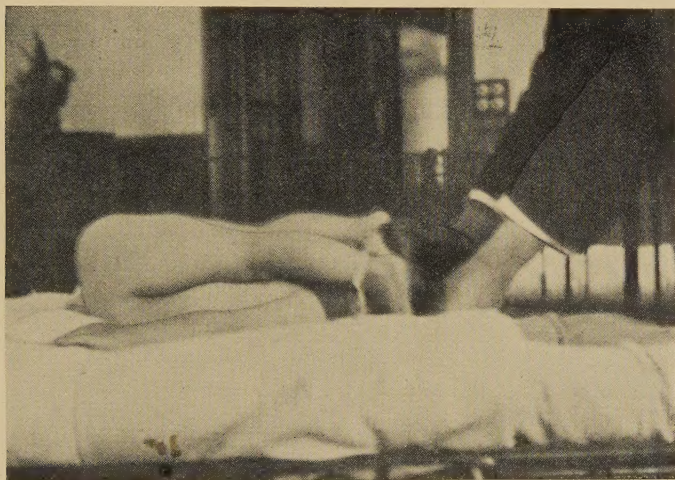


FIG. 2 (Case 1).—Shows child in "folded up" position.

Jumentié in the Société de Neurologie in Paris. The child had extreme hypotonia of the feet, and the limbs could be placed in abnormal positions. The arms, head, and trunk were also very hypotonic, even the tongue. The child showed choreic movements of the limbs. His speech was indistinct, but the intelligence seemed fairly good. There was no hypertonia of any kind. The Wassermann reaction was slightly positive.

The condition above described is not common, and the clinical picture is so different from that which occurs in most cases of cerebral diplegia and the pathological evidence is so meagre as to the cause of the condition that the subject is one which calls for further investigation. To the former we contribute some evidence with the hope that we may be able to deal with the latter subsequently.

CASE 1.—K. A—, aged two years, the second of two children, was born as a breech-presentation and weighed $3\frac{1}{2}$ lb. at birth. She has never walked, but got about on her buttocks.

On examination she is a well-nourished girl, can sit up well with straight back. She moves her arms and legs well. Her eyes and face show nothing abnormal. She cannot stand, and if any attempt is made to put her on her feet she extends them in front of her, but is unable to support the weight of the trunk. If held by the axillæ her legs go out in front flexed at the hip and extended at the knee (Fig. 1). If one tries to bend the legs in this position they are found to be rigid as in a spastic diplegia. When the child is lying in bed her limbs are found to be quite hypotonic. It is possible to "fold her up" (Fig. 2), her feet being flexed on the neck. The feet are very hypotonic and can be dorsiflexed on to the tibiæ. The knees can be hyper-extended. The knee-jerks and ankle-jerks are active. The plantar reflexes generally

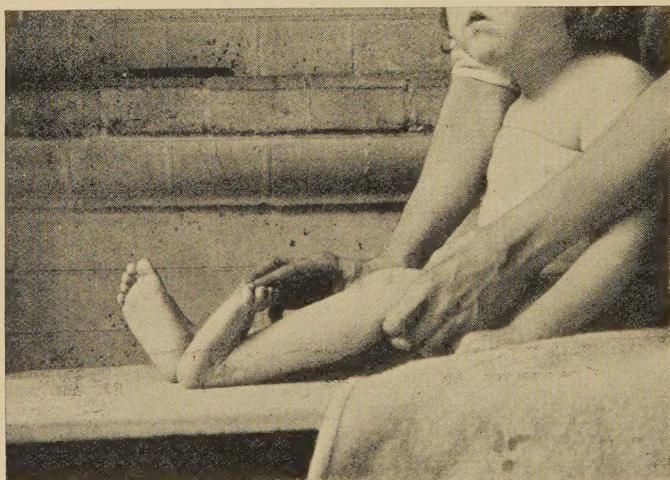


FIG. 3 (Case 2).—Shows the extreme hypotonia of the feet.

give a flexor response, but sometimes an extensor. The muscles respond normally to galvanism and faradism. There is no loss of sensation.

The child can only say a few words, but understands everything said to her. An X-ray photograph shows in addition a bilateral congenital dislocation of the hip-joints. An attempt was made to teach the child to walk by fixing the legs in an extended position in celluloid splints with the aid of a walking machine. But although the child made some attempt at progression she never could stand erect or walk by herself.

CASE 2.—C. H—, aged 3 years. The child has been weak from birth. He is the first of two children, born as a breech presentation after version. He has been clean in his habits since the age of eighteen months. He has never been able to stand or walk. On examination he is found to be a well-nourished child, can sit up and hold his head straight. There is a certain amount of kyphosis of the spine in the sitting position. He has difficulty in getting up because he cannot balance properly. Instead of lying down he drops down powerless according to gravity. There is marked hypotonia of the feet, which can be bent on to the tibiæ (Fig. 3.) There is hyper-extension of

the knee-joints and a certain amount of hyperflexion of the hip-joints. He cannot be "folded up," but can be placed in abnormal positions. There is a certain amount of flexor and adductor spasm (Fig. 4). When suspended by the axillæ his legs are extended and found to be rigid. There is marked hypotonia of the different joints of the arms. When lying in bed he can move his arms and legs freely, but all his movements show a great deal of inco-ordination. When placed on his feet he cannot stand unsupported, and when he attempts to walk his legs perform inco-ordinate movements. This ataxia is characterised by a lack of proportion of the movements. His favourite position is sitting on the side of his cot, supporting himself by the top railing, dangling his legs through the bars. The tendon reflexes are active. The abdominal reflexes show a marked overflow, so that the thigh muscles contract. The plantar reflexes give a flexor response. There is no loss of sensation, and the muscles

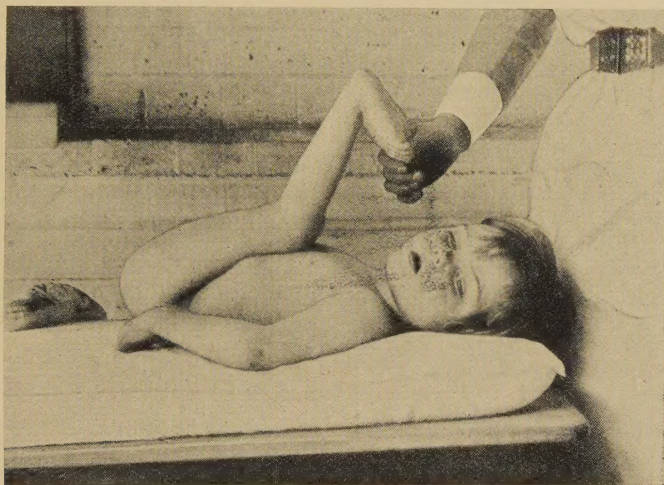


FIG. 4 (Case 2).—Shows the hypotonic foot with tone preserved in flexors of thigh.

respond normally to faradism and galvanism. The fundi are normal. He performs continuous sucking movements with his lips. There is nothing abnormal in the face. He seems fairly intelligent, but cannot talk. He can, however, say a few words without proper articulation. The Wassermann reaction is negative, and the cerebro-spinal fluid is normal. He is now taught to walk by fixing his limbs in a rigid position in celluloid splints. Though he makes attempts to progress with the aid of a walking machine there is much inco-ordination, not only of legs, but also of the trunk, which causes it to drop forward.

This child was formerly seen by Oppenheim, who considered it as a case of myatonia congenita.

CASE 3.—L. P—, a girl, aged 3 years, born as a breech presentation. She is the last of eleven children, six of whom died in infancy. She has had screaming and epileptic fits since the age of two years. She has never been able to stand or walk and never spoken a word. On examination she appears to be undersized and badly nourished. She is able to sit up by herself, and can remain in the sitting position, and holds her head straight. There is kyphosis of the spine in the sitting position. When

lying in bed she moves her arms and legs freely. There is marked hypotonia of the arms. The shoulders are loose, and the arms can be bent backwards to an extreme degree. The feet are very hypotonic and can be dorsiflexed to an extreme degree. There are hyperextension of the knee-joints and exaggeration of all the movements of the hip-joints. The child can be "folded up" to a certain extent, and there is no spasticity of any kind. The tendon reflexes are only with difficulty obtained. The plantar reflexes give a flexor response. When placed on her feet the child drops powerless down without making any attempt to stand or walk. She does not talk and is obviously mentally deficient. No changes of the fundi oculorum are found. The Wassermann reaction is negative, and the cerebro-spinal fluid is normal.

CASE 4.—D. L.—, a girl, aged 2 years and 10 months. She was a full-term baby, and the labour was quite normal. She is an only child. Nothing abnormal was noticed until the age of fourteen months, when the child started to have screaming fits. She has never been able to stand, walk, or talk. On examination she appears to be a well-nourished child. She is entirely mentally deficient. When lying in bed she throws her limbs about, and continually rubs the hands. She screams loudly when touched. There is an internal squint of the right eye, but no ocular palsy. The optic discs and maculae are normal. She has a high palate. She does not sit up by herself, but can remain in the sitting position when supported. She has loose shoulders and a marked hypotonia of the hands, which can be dorsiflexed to an exaggerated extent. She has a marked hypotonia of the legs, especially of the hip-joints. There is not much hypotonia of the feet and no spasticity. The knee-jerks are just present, weaker on the left side than on the right. The ankle-jerks are present; the plantar reflexes are indefinite. There is no inco-ordination. She cannot support the weight of her body, and there is complete inability to stand or walk. The muscles respond normally to galvanism and faradism. The Wassermann reaction in blood and cerebro-spinal fluid is negative, and the cerebro-spinal fluid on chemical and cytological examination shows no changes.

These four cases have in common a hypotonic condition of the voluntary muscles of the body, especially of the lower limbs, and a deficiency of the synergetic action of the muscles, as shown by the inability to stand or walk, but there is no loss of power in performance of active movements. One case shows a marked degree of ataxy, which is characterised by a lack of proportion of the movements (dysmetria of Thomas).

In two of the cases there is also a certain amount of hypertonia, as evidenced by the adductor spasm and overflow of the abdominal reflexes, in one case there is an occasional extensor response of the plantar reflexes. There also was in two cases rigidity of the lower limbs in a certain position, namely, when the children were suspended by the axillae.

Not one of our cases shows the extreme hypotonia described by Foerster with the exaggerated flexibility of the trunk, head, and neck.

Two of the children are markedly mentally deficient and mute, and their condition resembles closely that described by Pierce Clark.

It is interesting to note, however, that according to the statement of the parents these children have shown progressive degeneration.

Three of the children were born as breech presentation.

One showed another malformation, namely, congenital dislocation of the hips.

From the hypotonia associated with rickets these cases are easily distinguished by the absence of any characteristic rickety bone lesion.

The diagnosis from Oppenheim's myatonia congenita, which we consider as the simple atrophic form of the myopathies, remains entirely based on the condition of the tendon reflexes and the electrical reactions, which in the cases above described are normal.

The most interesting feature of this group of symptoms is the loss of tone and synergetic power associated with well-preserved active movement.

The pathology of this condition is practically unknown, and we may well suppose varies as much as the clinical picture of this and of the other forms of cerebral diplegia.

Edinger, in his summary "On the Cerebellum," comes to the conclusion that the maintenance of tonus is due to afferent impulses to the cerebellum from muscles, tendons, and from the labyrinth. They are transmitted by the nuclei of the medulla, pons, and mid-brain. Fibres of those centres are in connection with the basal ganglia. It is on a lesion of this system which includes the whole of the cerebellum and also is largely represented in the cerebral cortex (frontal lobes, temporal lobes, and motor region) that the pathology of this condition must be based.

Sherrington's "decerebrated rigidity" has recently been clinically demonstrated in the cases of aplasia of the cerebral hemispheres described by Sutherland and Paterson. There is good evidence to show that destruction of the cerebellum gives rise to loss of tone, though ablation of the cerebellum does not abolish the "decerebrated rigidity," and cerebellar lesions cause other symptoms as well as hypotonia.

Foerster, who expected a cerebellar lesion, found in two cases in which he obtained a post-mortem examination a sclerosis of the frontal lobes with macroscopically intact cerebella. There is no microscopic evidence, and there is nothing known of the conditions of the olives and the vestibular nuclei and the nuclei of the tegmentum.

We may be justified in supposing a gross cerebellar defect in the one of our cases with the cerebellar ataxia. It will depend on the

condition of the cerebrum how much this child will be able to improve. We know that interruption of the cerebro-cerebellar path or ablation of the frontal lobes exaggerates disturbances of the equilibrium caused by a simple cerebellar lesion.

In those cases where there is hypertonia as well as extensor response of the plantar reflex we will probably find that the pyramidal tracts are affected as well. No satisfactory explanation can be given why the hypertonia is brought on by the suspension of the children by the axillæ. It is a kind of reflex spasticity.

REFERENCES.

- (1) ANDRÉ-THOMAS.—"Cerebellar Functions," 'Nervous and Mental Disease Monograph Series,' No. 12, New York, 1912.
- (2) ANDRÉ-THOMAS and JUMENTIÉ.—'Rev. Neurolog.,' 1914, i, p. 566.
- (3) CLARK, L. PIERCE.—"Infantile Cerebro-cerebellar Diplegia of Flaccid Atonic-astatic Type," 'Am. Journ. Dis. Child.,' 1913, v, p. 425.
- (4) EDINGER, L.—"Ueber das Kleinhirn und den Statotonus," 'Deutsch. Zeitschr. f. Nervenheilk.,' 1912, xlv, p. 300.
- (5) FOERSTER, O.—"Der atonisch-astatische Typus der infantilen Cerebrallähmung," 'Deutsch. Arch. f. klin. Med.,' 1910, xcvi, p. 216.
- (6) SUTHERLAND, G. A., and PATERSON, H.—"On a Type of Cerebral Mal-development," 'Quart. Journ. of Med.,' 1913-14, vii, p. 61.

CARDIAC FAILURE IN A CHILD, AGED SIX YEARS, ASSOCIATED WITH ATHEROMA OF THE PUL- MONARY ARTERY AND EXTREME DILATATION OF THE RIGHT VENTRICLE.

By P. J. VEALE, M.B., B.S.Lond., Capt. R.A.M.C.,

AND

CAREY COOMBS, M.D., M.R.C.P.Lond.,
Assistant Physician, Bristol General Hospital.

ON May the 21st, 1913, a well-developed little girl, aged 5 years, was sent to the Bristol General Hospital by Dr. Newsome, of Pill. The history given by her mother was that, ever since an attack of bronchitis eighteen months before, she had been increasingly disabled by shortness of breath. No history of any other illness that could have had any causal relation with the condition which she now presented was forthcoming. Dr. Newsome told us that there was absolutely nothing in the family history to hint even faintly at a syphilitic factor. The child was very blue and obviously dyspnoic.

The fingers were not actually clubbed, but the nails were abnormally curved and the tips were shiny. No œdema was noted. The pulse was regular. Examination of the chest showed that the heart was enlarged to the left, the apex beat, which was wide and feeble, lying in the fourth left interspace a little way beyond the left mammary line. At this time no distinct enlargement to the right was made out. At, and external to, the point of maximum impulse a long, but not loud, systolic murmur was heard, and the pulmonic second sound was doubled. Neither at this time nor subsequently was any bruit heard at the base of the heart, though it was carefully looked for several times. At the bases of both lungs there were many alveolar *râles*, but the percussion note was not impaired. After a delay occasioned by the pressure of cases on the female medical beds, the child was admitted under Prof. J. Michell Clarke, whose notes (to which he has kindly allowed us to turn) describe a state of affairs identical with that noted above. The urine at this time contained a trace of albumin. It was also noted that the liver was palpably enlarged, but not the spleen. Unfortunately, after a stay of only one week she was taken home by her parents. The bronchitic signs were much less pronounced, but they had not disappeared.

On March the 25th, 1914, she was sent to the General Hospital once more, and admitted shortly after. The most striking feature of her case, apart from the dyspnœa and cyanosis, which were much more pronounced than on her last visit, was the supervention of œdema of the face, feet, and ankles. Her mother said this was variable, and certainly it diminished during the time she was waiting for a bed with a rapidity that was quite deceptive as an index of the general course of her case. When she came up the first time, on March the 25th, her face was so puffy that it seemed impossible to believe that she was not suffering from Bright's disease; however, there was only the smallest trace of albumin in the urine and no casts, though there were a few pus cells and many bacilli. The total daily output was small. There was a good deal of cough with expectoration of thick phlegm which, however, had no offensive smell. The fingers were no more clubbed than at her last visit. She also complained of pain in the stomach, increased by food, and of occasional vomiting. An examination of the chest discovered bronchitic signs much like those observed at the previous sojourn in hospital. The point of maximum impulse lay in the fifth left interspace, about one finger's breadth outside the nipple line; it was diffuse and not powerful. During expiration pulsation was palpable at the pulmonic area. A faint pulsatile movement could also be felt

to the right of the sternum. At, and external to, the point of maximum impulse a late systolic thrill was felt, and in the same area a long, definite, but not very loud bruit was heard, beginning late in systole and running up to and perhaps into the second sound. This bruit was conducted into the back by way of the left axilla, and was distinctly heard at the inferior angle of the left scapula. The pulmonic second sound was accentuated, but no murmur was heard at the base, or in the supraspinous fossæ behind, or in the neck. The cardiac dulness extended from the left anterior axillary line to a finger's breadth to the right of the sternum; upwards it reached to the inner end of the second left interspace. A skiagram showed very clearly that the cardiac volume was increased to the right of the sternum, and also upwards and to the left. During the days that intervened between her coming to the out-patient room and her admission to the wards, arrangements were made for taking an electrocardiogram, but the child, who lived at some distance from the hospital, was too ill to be brought up and back in one day. When she was admitted there was reason to think she had improved, for the bronchitic signs had disappeared and the œdema was very much less, but she was very listless and sleepy, and within two days after admission she died rather suddenly. Notes of the post-mortem examination, made on the same day, follow.

On opening the chest the heart was found to be considerably enlarged, owing to the dilatation of the right ventricle, which had come to occupy the whole anterior surface of the heart. On opening the heart the chamber of the left ventricle was found to be exceedingly small; the muscle wall was of good colour and fairly firm and measured 1 cm. in thickness. The left auricle similarly was of small size with thickened walls.

The cavity of the right ventricle was greatly dilated (holding approximately 4 oz.), with considerable hypertrophy of the moderator band and all the papillary muscles, which also showed fibrous thickening of the subendothelial tissues. The right auricular cavity was dilated, the wall measuring 5 mm. in thickness.

The foramen ovale was closed except for a pin-point orifice at the inferior border. There was no opening in the undefended space of the septum, and the ductus arteriosus was represented by a fibrous cord only. The mitral opening was normal in size; its aortic cusp showed some atheromatous patches, particularly on its ventricular aspect, and the vessels at the root of the valve flaps were congested. The aortic orifice admitted the little finger; the valve segments were somewhat clouded and thick, and had small spots of atheroma at

the junctions of the lunules, whilst some recent atheroma was seen along the margins of the pockets, and at the insertion of the cusps into the root of the aorta. The tricuspid valve admitted two fingers, its margins were firm and thickened, and the endothelium on this surface of the valves was transformed into a dense white membrane with subendothelial nodosities here and there.

The pulmonary orifice measured 15 mm.; the valve segments showed no sign of disease. In the descending aorta were long streaks of atheroma along its length, particularly surrounding the orifices of its branches. The pulmonary artery and its branches were the seat of a very pronounced nodose atheroma.

Microscopically the atheromatous patches showed great thickening and multiplication by lamination of the endothelial tissues; the internal elastic tissues and loose subendothelial elastic fibres were replaced by a highly cellular loose areolar tissue, the cells being round cells with a reticular nucleus and a nucleolus. Very few fibroblasts were to be seen. Beneath this cellular layer there was a well-organised tissue composed of fibres loosely arranged, but in two or three patches aggregated into a dense nodule resembling a scar. There was no cellular infiltration of the deep elastic layer until the line of demarcation between that layer and the muscle tissue was reached, when a very obvious round-celled infiltration was observed. The capillaries in the muscle layer showed a very marked endothelial proliferation, as did those within the elastic layer. There was no evidence of acute gummata, nor were any treponemata to be found in sections stained with silver.

The muscle of the ventricle and auricle appeared normal; there was no infiltration of the connective tissue or any focal areas of cellular aggregation, and the blood-vessel walls showed no sign of any disease. There was no evidence of disease in the mediastinal glands.

COMMENTS.

(1) *Ætiology*.—Atheroma of the pulmonary artery has been encountered under two sets of circumstances. As an accompaniment of bronchitis, emphysema, and disease of the mitral valve, it is not very rare. In such cases it is ascribed to hypertension in the pulmonary artery, secondary to the disease of heart or lung. In another much smaller group of cases the arterial lesion appears to be primary, *i. e.* it is not associated with any intra-thoracic disorder that can be held responsible for its development. It is in this second group that we place our case; for the bronchitic phenomena

that were present during life were mild in degree, and were proved by post-mortem examination to have been due to passive hyperæmia. They were an effect of the cardiac lesion, and not its cause. Possibly some of the cases in which pulmonary atheroma has been regarded as a sequela of bronchitis are also of this order; for instance, in an old post-mortem book at the Bristol General Hospital, we have come across the case of a man, aged 23 years, to which the diagnosis of "bronchitis" was attached, in the autopsy as well as in the clinical notes; yet there is no doubt, from the pathological data given, that atheroma of the pulmonary artery and dilatation of the right heart were the conspicuous changes.

An excellent summary of the whole subject is to be found in a monograph by Posselt published in 1908. From this it would seem that though the number of cases in which the atheroma was apparently primary is small (13), yet the average age of such cases (39) is lower than that at which serious atheromatous changes are commonly noted in the aorta and systemic arteries. Since this was written Sir Leonard Rogers, of Calcutta, has published an account of ten similar cases occurring in Bengal, which show that in that district primary atheroma of the pulmonary artery is not so rare as it is in Europe. In his cases also the average age was only 32. The youngest patient was 12, and we can find no record of any younger. Rogers surmises that syphilis may have been responsible for some of his cases, but in our patient this was fairly excluded, not only on clinical grounds, but also by the absence of spirochætes from the arterial lesions and by the lack of any post-mortem evidence of syphilitic infection. Posselt, in his monograph, lays stress on the importance of severe infections, particularly variola, in earlier life, but in our patient there was no hint of any such factor, apart from the presence of *B. coli* in the urine. It should be added that in a few old people the pulmonary arteries share in the sclerotic process which has also invaded the systemic vessels.

(2) *Pathology*.—An interesting problem is the relation between pulmonary atheroma and failure of the right ventricle. Though the musculature of the right ventricle showed but little microscopical alteration, this does not negative the view that its functions were profoundly affected; the disproportion between interference with myocardial function and perceptible alteration of myocardial structure is a commonplace of cardiac pathology. The fact that the right heart was enormously dilated, out of all proportion to the enlargement of the left side, affords tangible proof of the extent to which its musculature was injured. The question that one naturally asks is

—Did the arterial lesion cause the ventricle to dilate, or did both phenomena own some common origin? To this question our case gives no sort of answer, for there was no discoverable cause for the arterial disease or for the ventricular dilatation apart from the arterial lesion.

(3) *Symptoms*.—In our case the most striking clinical feature was deep cyanosis. This seems to have been noted in many of the cases that fall within the heading of primary pulmonary atheroma. Second to this comes œdema, also noted in a majority of the recorded cases. In many of these it was, as in our case, a late phenomenon. In our case the swelling was so great and so general that it seemed almost incredible that the urine should be normal, but it is quite certain, on post-mortem as well as clinical evidence, that there was no nephritic factor in the case. The physical signs differed widely in the reported cases; in several the diagnosis made during life was that of congenital malformation of the heart, while in others an acquired lesion of the mitral valve was suspected. We suggest that the coincidence of cyanosis and œdema with evidences of enlargement of the right heart, and absence of the characteristic signs of congenital malformation or of acquired mitral disease, might point to a diagnosis of pulmonary atheroma. Rogers was actually enabled to make the diagnosis in one of his cases. We lay stress on the skiagraphic method of discovering enlargement of the right heart, since it was very serviceable in our own case. It is possible that a condition of this kind might reveal itself by some characteristic form of electrocardiogram; in our case the child grew worse so rapidly, and died so soon after admission, that we were unable to take a record.

REFERENCES.

- (1) POSSELT.—‘Samml. Klin. Vorträge’; ‘Innere Med.,’ No. 149/52, Leipzig, 1908.
- (2) ROGERS.—‘Quart. Journ. Med.,’ Oxford, 1908–9, ii, p. 1.

HÆMATOMA OF THE LOWER LID IN AN INFANT, AGED 5 MONTHS: AN UNUSUAL MANIFESTATION OF INFANTILE SCURVY.*

By SYDNEY STEPHENSON,

Ophthalmic Surgeon to the Queen's Hospital for Children, London.

A FEMALE infant, aged 5 months, was brought to the Eye Department

* The case was reported to the Section for the Study of Disease in Children of the Royal Society of Medicine on January the 22nd, 1915.

of the Queen's Hospital for Children, London, on January the 7th, 1915.

The history of the case was to the effect that the infant did well until the middle of last December, when she developed "thrush," diarrhœa, and vomiting, symptoms from which she made a fair recovery in the course of about a fortnight. On January the 3rd a small black spot or line was noticed in the skin of the right lower eyelid. There had been no injury to the parts. No especial importance was attached to the mark; but on the following morning the eyelid was swollen and discoloured, and since then no particular change had been noticed in the condition of the parts. On January the 6th, the day before the infant attended the hospital, the medical practitioner under whom the patient had been made a puncture into the distended lower lid, with the escape of what was described as "blood and matter."

As to diet, for the first two months of life the baby was breast fed, and after that Nestlé's milk was given exclusively. Since recovery from the attack of December last nothing has been given in the way of food except albumin water. No patent foods have been used in bringing up the infant.

On admission, the right eye was completely closed by the pressure of the lower lid, which was enormously distended with blood. The swollen parts were almost chocolate-brown in colour. A recent puncture, exuding a little blood, was present at about the junction of the outer third with the inner two-thirds of the lower lid. The upper eyelid was not swollen, and exhibited merely a few insignificant patches of cutaneous ecchymosis. The eyeball was not protruded. The cornea was clear. Temperature 99° F. The child had some colour in her cheeks, and although somewhat wasted (weight, 11 lb. 6 oz.) yet looked tolerably well. No tenderness on handling. No swellings about legs or arms. There were no teeth, and the gums were in excellent condition.

A provisional diagnosis of infantile scurvy was made, and the child admitted as an in-patient. She was placed on equal parts of "raw" milk and water. One teaspoonful of raw meat juice was given every three hours, alternatively with a similar quantity of orange juice. The affected eyelid was treated with compresses of hydrogen peroxide.

The improvement in the state of the eyelid was striking. Two days after admission the condition was less marked, and the puncture mentioned above was healed. A couple of days later the baby could open the eye somewhat, and seven days after admission the eye could be widely opened, and the lower lid, although blood-stained like a "black eye," was not swollen. In fact, in the course of a single

week under anti-scorbutic diet changes had taken place that in ordinary cases would probably have spread over three weeks or a month.

The infant's general health, unfortunately, did not improve *pari passu* with the local condition. It is true that during the week's stay in hospital the child gained 3 oz. in weight, but on January the 8th, the day after admission, the pulse was 172 per minute, and the temperature 102·8° F. Since then the temperature has not fallen below 101·4° F., and has generally been between 103° F. and 104° F., without any definite cause having yet been found to account for it. The stools, which have numbered from three to seven a day, were found to contain blood on January the 12th and 13th. On January the 14th the urine contained a trace of blood.

On January the 16th signs of meningitis developed, and the child died on January the 18th. Post mortem pneumonia of both lungs and pneumococcal meningitis were found.

Comments.—The foregoing, I think, can only be regarded as an instance of infantile scurvy with unusual ocular manifestations. Apart from the difficulty of accounting for a non-traumatic hæmatoma in so young a child on any other view, there is a certain amount of evidence in support of the suggestion: (1) The extraordinarily rapid subsidence of the effused blood under an anti-scorbutic diet, and (2) the discovery of blood in (a) the stools and (b) the urine. On the other hand, the classic signs of infantile scurvy were conspicuous by their absence. The case is further peculiar in that the ocular manifestations preceded any other signs of scorbutus. For example, melæna did not develop until nine days, and blood in the urine until eleven days, after the eye signs. In this point the case recalls one reported by W. T. Holmes Spicer* in an infant, aged 7 months, who was brought because the right upper eyelid was tensely swollen and blood-stained. There were no other manifestations of infantile scurvy. Rapid improvement took place once the child was placed on suitable diet, together with cod-liver oil. A case analogous with my own has been placed on record by K. Steindorff† in a child, aged 7 months, whose left upper eyelid was greatly distended by blood and of a bluish-red colour. Lesions of the gums were present. The child's body was exquisitely tender when touched. Traces of blood were found in the urine. One small conjunctival ecchymosis was present, but there were no retinal changes.

* 'Trans. Ophthal. Soc. U.K.,' 1892, xii, p. 33.

† 'Zeitschr. f. Augenheilk.,' 1911, xxv, p. 180.

PNEUMOCOCCUS INFECTION AND IMMUNITY.*

By RUFUS I. COLE, M.D.

MOST of the cases of acute lobar pneumonia occurring in all parts of the world are apparently caused by *Diplococcus pneumoniae*. Certain observations, however, indicate that all these cases that are apparently due to pneumococci do not have an identical ætiology. Certain ones are due to *Pneumococcus mucosus*, first described by Schottmüller as *Streptococcus mucosus*. Moreover, on the basis of immunological differences, it is possible to classify the other pneumococci in three groups. In two of these groups, which we have numbered Types I and II, have been placed organisms having immunological characters peculiar to the respective group. All the organisms belonging to Type I, for instance, are agglutinated by a serum produced by the immunisation of an animal to any one of them, and such an immune serum also protects mice against any one of the group. In Group III are placed all the organisms of the *Pneumococcus mucosus* type. These organisms are not agglutinable, and an immune serum does not protect mice against them. In addition to the organisms belonging to these three groups, however, there are a number of organisms which have no common immunological characters; each one appears distinct. These organisms have been grouped together as Group IV, or Type IV.

Cultures received from Prof. Neufeld in Germany indicate that the same types of organism are prevalent in Germany as are found here, and studies made by means of our immune serum by Dr. Walker in Boston, and Dr. Lewis at Philadelphia, indicate that similar organisms are found in cases of pneumonia in those cities. In 150 cases in which the type of organism causing the disease was determined, 38 per cent. proved to be due to organisms of Type I, 30 per cent. due to organisms of Type II, 11 per cent. due to organisms of Type III, and 21 per cent. to organisms of Type IV. About the same relative frequency of occurrence of the different types has been found in Philadelphia by Dr. Lewis. Apart from the organisms of Type III, there are no constant fixed morphological or cultural types among these different races. The clinical manifestations of infection due to these different types, however, are not identical certainly as regards severity. Among 103 cases not receiving specific treatment, the mortality among those due to organisms of Type I was 25 per cent.;

* The Frederick A. Packard lecture delivered before the Philadelphia Pediatric Society on November the 10th, 1914.

among those due to organisms of Type II, 32 per cent.; among those due to organisms of Type III, 47 per cent.; and among those due to Type IV, 6 per cent. From the standpoint of prognosis alone, therefore, the determination of type of infection in the individual case is of considerable importance. The conditions in pneumonia are only different in degree from those found in typhoid fever and dysentery, where infections due to organisms of different types occur.

Studies made by Dochez indicate that the type of organism commonly found in normal mouths is that which we have called Type IV. In two instances, however, in the mouths of persons very closely associated with pneumonia patients, pneumococci of the fixed types have been present. Dr. Lyall has shown that the pneumococci commonly present in the mouths of patients suffering from tuberculosis are also of Group IV, and in only two or three instances were pneumococci of the fixed types present. In the mouths of patients convalescing from pneumonia pneumococci of the fixed types usually disappear within a few weeks after crisis, and are replaced by organisms of the fourth group.

These studies indicate that it may be justifiable to consider all persons having pneumococci of the fixed types in the mouth as carriers of infection. The facts, however, are still too few to justify conclusions as to the best practical measures to be taken for the prevention of pneumonia. Certain observers have apparently observed the transformation of one type of pneumococcus into another—even pneumococci into streptococci, and *vice versa*. In our own personal experience, however, we have not seen such a transformation of one type into another. A study by Dr. Chickering of the agglutinins present in the blood of patients suffering from pneumonia also indicates that the type of organism in any given case does not change—certainly after the infection has actually commenced. Until the evidence in regard to transformation is more definite, it does not seem necessary to take it into too serious account in our consideration of the epidemiology of pneumonia.

Intoxication.—It has so far been impossible to detect by chemical or biological means the presence of poisons, either in the media in which pneumococci have been cultivated, or in the body fluids of animals dying from infection with these organisms. Lately, considerable stress has been laid on the importance of bacterial anaphylatoxins, as described by Friedberger, in producing the symptoms of infection. The anaphylatoxin theory assumes that the intoxication is due to split products of the bacterial protein. According to Friedberger the ferment causing the splitting is supposed to be

present in the serum, but the demonstration by Neufeld and Dold, and also by Rosenow, that autolysed bacteria, in the absence of serum, are toxic has required that the theory be modified so as to presuppose that the ferments are present in the bacterial bodies. We have been able to show, however, that the bacteria simply dissolved in dilute solutions of bile, or even brought into solution by freezing and grinding, are also toxic. Since, in the latter experiment, digestive processes can with all probability be excluded, it seems reasonable to believe that the toxic effects produced by these various solutions are due to preformed substances within the bacterial cells, which are set free when the bacteria go into solution. We have also shown that such solutions of bacterial cells are hæmolytic. By immunisation of animals it has been possible to produce a serum which neutralises the hæmolytic effects of the toxin, but, so far, it has been impossible to obtain a serum which neutralises the toxic effects.

The evidence is still very inconclusive, however, that either one of these reactions is of significance so far as intoxication in lobar pneumonia is concerned. The experimental evidence indicates that the symptoms are due to the action of the living bacteria, rather than to the action of substances contained within the dead bacterial cells. Pneumococci, when growing in blood-containing medium, or when growing within the animal body, are able to transform oxyhæmoglobin into methæmoglobin. An analytic study of this reaction has suggested a possible way in which pneumococci may produce their effects without the production of a soluble poison. This study indicates that the transformation of oxyhæmoglobin into methæmoglobin is due to a modification of oxydation and reduction processes in the medium immediately surrounding the bacteria. If this be true, it is possible that an analogous reaction may be responsible for the toxic action of pneumococci on tissue cells. A difficulty, however, arises in applying this theory to pneumonia, since early in the disease there is apparently no wide distribution of the organisms. It is conceivable that in the early stages the symptoms are non-specific in character, and only in the later stages of the disease, when the pneumococci are widespread throughout the body, is the intoxication truly specific in nature.

In the last analysis, however, the exact nature of the intoxication in pneumonia is still obscure.

METHOD OF SPECIFIC TREATMENT.

The method of specific treatment of bacterial infection for which there exists, at present, the best experimental justification is by

means of specific immune serum, passive immunisation. The diseases in which the best results from this form of treatment have been obtained are diphtheria and tetanus. The serum employed in these diseases is antitoxic. It is for this reason that we have made such great efforts to determine the nature of the intoxication in this disease. In spite of the fact that the evidence is not very strong that the toxic substances isolated by solution of the bacterial cells play any great rôle in pneumonia, we have, nevertheless, attempted to produce immune serum to such substances. As previously stated, however, such an immune serum has very slight antitoxic power as tested in guinea-pigs, and it has also been found to have only slight protective action when injected together with living organisms. The efforts, therefore, to produce an antitoxic serum which would be of use in the treatment of this disease have so far been unavailing. On the other hand, that it is possible to produce immune sera having high protective power against pneumococci by the injection of repeated increasing doses of the living bacteria has been known for a long time. Immune sera produced in this way have in fact been largely employed in the past in the treatment of pneumonia, but the clinical results obtained have not justified their continued extended employment. Further experimental studies, however, have disclosed several probable reasons why such immune serum has been of little or no value. In the first place, a pneumococcus immune serum is effective only against the type of organism used in the process of immunisation. If the infection is due to an organism of Type I, for instance, only an immune serum produced by the injection of organisms of Type I, will be of any value. In order to render an anti-pneumococcus serum useful in any type of pneumonia it would be necessary first to produce a serum with high protective power against the type of organism concerned; second, to have a satisfactory method of standardisation of such serum; and lastly to have a method for quickly determining the type of organism in each individual case. These conditions have now been satisfactorily met, as far as organisms of Type I and Type II are concerned. As regards the organisms of Type III, while high active immunity can be produced against them, the serum of such actively immunised animals is entirely without power of preventing infection of other animals. As before stated, each organism of Group IV seems to be absolutely distinct, so that treatment of cases due to this type of organism would be practically impossible.

There are two additional reasons why the employment of immune serum as it was carried out in the past was not likely to produce

favourable results. First, it was not used in sufficiently large amounts, and second, its employment was generally made too late in the disease. On the basis of experimental evidence so far obtained, it seems that if treatment be given in large amounts, and not too late in the disease, the use of immune serum against organisms of Types I and II should be useful in the cases of pneumonia due to these organisms respectively. It has been found possible to produce a serum of great protective power against organisms of Type I and Type II. In the clinical studies so far carried out, the serum treatment of cases due to organisms of Type I has yielded more promising results than the serum treatment of cases due to organisms of Type II. Judgment as to the value of the serum has not been based on mortality statistics alone, but the following two facts are considered as evidence of the value of the serum. First, where the blood cultures have been positive before beginning treatment, after one dose of serum, and before the second was given, cultures from the blood have been sterile. Second, it has been found that in the treated cases the appearance of immune substances in the blood occurs much earlier than it does when no such specific treatment has been administered. While the employment of large doses of serum such as we have used is usually followed by so-called serum sickness, this, in future, will undoubtedly be avoided to a large extent by the employment of concentrated serum.

Stress has been laid on the work carried out in the hope of finding a rational basis for serum-therapy, rather than on the actual practical results already obtained. It is only by discovering the underlying principles that we can hope to obtain satisfactory measures for combating this terrible infection. We can only hope that careful experimental studies have given us justification for the employment of immune serum in the way I have mentioned, and that the practical results on a large series of cases may confirm the conclusions drawn and the results already obtained.

Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, January the 22nd, 1915.

The President, MR. T. H. KELLOCK, in the Chair.

Case of Congenital Syphilis.—Dr. F. D. SANER showed a girl, aged 9 years, who had been apparently quite healthy until just over two years ago,

when she had some pain and stiffness in the left elbow and ankle-joint. Some months later a small hard lump appeared about the centre of the forehead, which gradually enlarged and finally ulcerated. The left leg became very deformed, and for nine months before admission the child was unable to walk.

No specific treatment had been given until the child was admitted to hospital on November the 11th, 1914. She was then extremely wasted and anæmic, did not eat or sleep. There was a large, circular, foul-smelling ulcer, with a sloughy base, in the centre of the forehead, quite half as large again as the present condition. The eyes were almost closed from œdema of the upper lids. The liver and spleen were distinctly palpable. Wassermann's reaction was positive. Since admission hyd. c. creta 1 gr. had been given three times a day, and an inunction of ung. hydrarg. $\frac{1}{2}$ dr. once daily. In addition, pot. iod. 5 gr. c. inf. gentian co. ad. 2 dr., *t.d.s.*, has been given, and sanitas fomentations to the forehead. A hydrogen peroxide mouthwash had been used, and the treatment has been very well borne.

The child had gained 12 lb. in weight since admission, and her general condition was satisfactory.

Case of Enlarged Liver in a Boy, aged 15 years.—Dr. H. W. BARBER and Dr. F. D. SANER showed a boy, aged 15 years, admitted to hospital on December the 27th, 1914, for pain in the right side, which had only been present for a few days. The boy was of average development for his age, and there is no history of any previous illnesses. The liver was considerably enlarged; on the right side its lower border nearly reached the iliac crest; in the mid-line its margin was just below the umbilicus, and it could be traced to the left costal margin at the level of the ninth costal cartilage. The spleen was not enlarged. The Wassermann reaction was positive. There was no eosinophilia.

Hæmatoma of the Lower Lid in an Infant, aged 5 months: an unusual Manifestation of Infantile Scurvy.—Mr. SYDNEY STEPHENSON (*v. p. 77*).

Polio-encephalitis with Ocular Manifestations only.—Mr. SYDNEY STEPHENSON and Dr. SYDNEY A. OWEN showed a case in a girl, aged 8 years. She had gone to bed in her usual health, but the next morning the eye was turned, and she complained of pain over her right brow. Examination three days later showed slight ptosis of the right eye, which was directed outwards and upwards. There was a defect in the downward and inward excursions of the eye. The secondary deviation was outwards and a little downwards. Diplopia was crossed, and the false image lay at a lower level than the true, its upper end inclined outwards. The signs therefore indicated paresis of the right inferior rectus. The right pupil measured 6.5 mm. and responded sluggishly to light. The left measured 5 mm. and was fairly active. There were no other signs of nervous disease.

Infantile Paralysis with Excessive Knee-jerk.—Dr. SHEFFIELD NEAVE showed a boy, aged 2 years. The right leg presented flaccid paralysis, deficient response to electrical stimuli, wasting of the calf-muscles, excessive knee-jerk, and extensive plantar response.

Congenital Heart Disease and Ulcerative Sore Throat.—Dr. J. D. ROLLESTON.—A male infant, aged 11 months, was admitted to hospital on

December the 6th, 1914, certified to be suffering from diphtheria, on the eleventh day of disease. The father had always been healthy, but the mother had been an in-patient at the Brompton Hospital on several occasions for rheumatism with cardiac complications. The child had always been delicate and blue from birth. Clubbing of the fingers had been noticed since he was aged 2 months.

On admission the child appeared very ill. There were membranes on the tonsils, pillar, and uvula, and a profuse nasal discharge. The heart sounds were clear but rapid. There was a trace of albumin in the urine. A large dose of antitoxin was given on admission and on the following day. A few organisms resembling diphtheria bacilli were found in the cultures, but cocci were predominant.

On December the 8th a loud systolic murmur was audible all over the præcordium. There was marked ulceration of both tonsils. No Vincent's organisms were found in the throat smears. The ulceration gradually spread over the uvula and palate.

On December the 13th stridor and a hoarse cough developed, and there was much difficulty in swallowing. The symptoms persisted, and the general condition became worse until death took place from bronchopneumonia on December the 20th. The heart showed the following anomalies:

(1) Transposition of the great arterial stems, the aorta arising from the infundibulum of the right ventricle, and the pulmonary artery from the left ventricle.

(2) Marked deficiency of the interauricular septum, which was represented by a narrow band 1.5 cm. long by 0.5 cm. broad. The foramen primum lay anterior to it, and posteriorly there was a widely patent foramen ovale.

(3) An intraventricular foramen, 1.2 cm. in diameter, in the "undefended space."

(4) Stenosis and hypoplasia of the pulmonary artery, the valve of which had only two cusps.

The fauces and larynx showed ulceration of the tonsils, frenum, epiglottis, right aryteno-epiglottidean fold, and deep ulceration of the laryngeal portion of the pharynx on each side, especially on the left, exposing the muscular tissue and eroding the thyroid cartilage.

Acute Atrophy of the Liver.—Dr. J. PORTER PARKINSON showed a specimen from a girl, aged $5\frac{1}{2}$ years, who had had jaundice for five weeks. At first the liver was enlarged and then gradually got smaller, till it could no longer be felt. The urine contained bile pigment and acetone, but no albumen, leucin, or tyrosin. Delirium set in and death took place. Post mortem the liver weighed $1\frac{1}{4}$ lb., was pale yellow and very friable. On section it showed yellow areas, between which were small red spots. The gall-bladder and ducts were normal. Microscopically there was general staining with bile pigment, the cells were distended with fat, but the nuclei were well marked. There was no distension of the bile-ducts and no actual necrosis of the cells. No micro-organisms were found in the specimens.

Suprarenal Hæmorrhage in an Infant.—Dr. E. B. GUNSON showed specimens from a case of suprarenal hæmorrhage in an infant. The child was first seen on the sixth day after birth. There was a slight discharge from the umbilicus, the cord having separated. On the eighth day the temperature rose suddenly to 102° F., and cellulitis of the forearms developed. The

stools became frequent, loose, and offensive. The child was collapsed and died on the ninth day, thirty hours after the onset of symptoms.

Post mortem the right suprarenal was greatly enlarged, and on section was found to have been converted into an almost black hæmorrhagic pulp. The left suprarenal was similarly affected, but to a less extent. A culture from the heart's blood gave a pure growth of the streptococcus; a Gram-stained perforation of the left suprarenal showed the presence of streptococci both in the cortex and medulla.

Purpura complicating Diphtheria.—Dr. E. B. GUNSON also showed a photograph of this condition in a girl, aged 10 years. The purpura appeared at the site of an antitoxin rash and persisted till death. Before death the purpuric patches on the cheeks and ears became confluent. Superficial ulceration occurred, but there was very little loss of substance. Hæmorrhages occurred into the gums, and an extensive subconjunctival hæmorrhage appeared in the right eye.

There was old-standing constipation, the stools resulting from the administration of castor oil and turpentine being very copious and markedly offensive for many days.

Palatal palsy, carditis with "cardiac paralysis," arthritis, and albuminuria developed before death on the twenty-eighth day.

Teratoma of Pharynx.—Dr. F. J. POYNTON and Mr. T. T. HIGGINS showed specimens illustrating a persistent pharyngeal rudiment in an infant, aged 5 months. Some difficulty in swallowing, associated with coughing and occasional dyspnoea, had been noticed for five weeks. A pearly white, rounded, polypoid tumour was discovered in the pharynx, and under anæsthesia was found to spring from the left supratonsillar fossa, to which it was attached by a narrow fibrous stalk. After removal it was found to contain a narrow band of cartilage, embedded in fibrous connective tissue, and covered by perfectly formed skin.

Abstracts from Current Literature.

Medicine.

Organic acids in the urine of infants (*Monatsschr. f. Kinderheilk.*, 1914, XII, p. 645).—H. ARON and MARIANNE FRANZ consider the lowest members of the fatty acid series (formic, acetic, and butyric acids) and oxalic acid, and as a result of their experiments they find that only very small quantities of volatile fatty acids are excreted in the urine of infants, that food rich in fat does not affect the quantity of the acids, and in acute disturbances of nutrition the quantity of volatile acids is not increased in the urine. As regards oxalic acid, they found that with a mixed diet (mother's milk, cow's milk, or cow's milk and sugar) free from oxalic acid, the urine almost invariably contained small quantities of oxalic acid which did not arise either in the bowel (result of fermentation) or from intermediate metabolism (endogenous oxalic acid formation). The quantity of oxalic acid in the urine was somewhat higher with the administration of cane or grape sugar than with a pure milk diet. Barley meal increased the excretion of uric acid markedly. There was no increase of uric acid excretion in the urine in acute disturbances of nutrition.

F. R. B. ATKINSON.

The presence of lactic acid in the urine in cyclic vomiting ('*Am. Journ. Dis. Child.*,' 1914, VIII, p. 127).—**F. P. Underhill** and **H. M. Steele** record a case of cyclic vomiting in a boy, aged $2\frac{1}{2}$ years, from whose urine more than .56 gm. of zinc lactate was isolated in the twenty-four hours. No lactic acid was found in the urine in the intervals between the attacks. Diacetic acid, acetone, and protein were also present, gradually disappearing as the attacks passed off. The significance of lactic acid in the urine is obscure.

J. D. ROLLESTON.

Some experiments in connection with orthostatic albuminuria ('*Practitioner*,' 1914, xciii, p. 113).—**F. D. Nicholson** examined the urines of 189 boys in Framlingham College on two different dates, and found 28 per cent. had albuminuria. The tests were made at 7 a.m. after dressing, 10 a.m. after breakfast, 3.30 p.m. after football, at 10.30 a.m. and at 3 p.m. after a three-mile run. Seven per cent. showed albumin after getting up, 10.7 per cent. after football, and 18 per cent. after the run. The urines of forty-two boys were examined just before and after a plunge in the swimming bath. Four passed albumin at both times, whilst in three the test was positive only after the swim.

F. R. B. ATKINSON.

Acute nephritis in infancy from disturbances of nutrition ('*Arch. f. Kinderheilk.*,' 1914, LXXIII, p. 202).—**F. Frank** records fourteen cases in children, aged from 1 to 11 months. His conclusions are as follows: (1) Acute nephritis is not frequent in infancy. (2) It is pre-eminently of an exudative nature and frequently has an hæmorrhagic character, which is to be attributed to the excessive permeability of the blood-vessels in the first year of life. (3) Ætiologically all kinds of infections play a part and especially disturbances of nutrition. (4) Among the varieties of nephritis associated with disturbances of nutrition the ascending form must be assigned a special significance, since the lower urinary passages are frequently involved in infantile digestive disturbances.

J. D. ROLLESTON.

Mild forms of chronic infantile nephritis ('*Gazz. med. ital.*,' 1914, LXV, p. 77).—**V. Stavsky** draws attention to chronic intermittent albuminuria in childhood, the patients presenting no symptoms except perhaps slight anæmia, headache, and puffiness of the face. The condition is allied to that described by Heubner under the name of *pædonephritis levis*. A characteristic of the affection is the occurrence from time to time of slight hæmorrhages almost always in connection with mild acute infective diseases of other organs, especially the tonsils. As regards ætiology, it is interesting to notice that it occurs almost exclusively in late childhood. In Heubner's thirteen cases there was only one, a girl, aged 3 years, all the others were more than six years old, and seven of them were ten to twelve years old. The female sex predominated, there being nine girls and four boys. From a diagnostic point of view it must be borne in mind that the true nature of the complaint may be marked by manifestations of anæmia, and on this point two cases reported by the author are particularly instructive. One was a boy, aged 10 years, who, after an attack of scarlet fever three months previously, continued in feeble health, complaining of headache, nausea, and anorexia. No organic disease could be found except that the urine contained albumin (0.5 per mil.), hyaline casts, and white corpuscles. Subsequent examination proved the albumin to be permanent, oscillating from 0.5 to 0.8 per mil. This was the only case known to the author where a mild

nephritis became transformed into a severe form. If these cases of nephritis may get completely well, it must be recognised that treatment has little effect; the administration of arsenic, however, should be avoided; the author considers it has a deleterious action on the kidneys, and it should never be given in cases of anæmia without ascertaining first that a mild form of chronic nephritis is not present.

VINCENT DICKINSON.

Pyelitis in infancy and childhood (*Boston Med. and Surg. Journ.*, 1914, CLXX, p. 540).—**E. T. Wyman** analyses a series of sixty-five cases at the Boston Children's Hospital. Fifty-four per cent. were in infants under two years, and 46 per cent. in older children. Sixty-three per cent. of the infants were females, and 90 per cent. of the children were females. Chills occurred in 15 per cent., dysuria in 36 per cent., frequent micturition in 40 per cent., vomiting in 39 per cent. The leucocyte count varied from 8000 in a chronic case to 62,000 in a very acute case; average of all uncomplicated cases 19,000. The organism responsible was the colon bacillus in a large proportion of the cases; streptococci, staphylococci, and pneumococci were sometimes found.

REGINALD MILLER.

Bacillus coli infections in infants and early life (*Med. Record*, 1914, LXXXVI, p. 288).—**W. M. Hartshorn** records two cases of protracted *Bacillus coli* pyelitis in a female, aged 20 months, and a male, aged 6 months. The latter had slight stiffness of the neck and tendency to opisthotonos, followed by a condition of tetany which lasted two months. Lumbar puncture was negative. Treatment consisted in the administration of large doses of potassium citrate and the guarded use of urotropin in increasing doses. Vaccines were tried in one case, but were followed by an aggravation of the symptoms.

J. D. ROLLESTON.

Circumcision in the masturbation of female infants (*Am. Journ. Dis. Child.*, 1914, VIII, p. 144).—**R. J. Freeman**, after alluding to Rachford's paper on masturbation in infants (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1907, IV, p. 510), states that the habit is due to the irritation caused in nervous children by adhesions between the clitoris and the surrounding tissue. The only curative treatment consists in separation of the adhesions and circumcision. Six illustrative cases are recorded.

J. D. ROLLESTON.

Vulvo-vaginitis in children (*Arch. de Méd. des Enf.*, 1914, XVII, p. 209).—**J. Comby** refers to a series of cases of vulvo-vaginitis which he published in 1891. This series comprised 152 cases, varying in age from three weeks to fifteen years, but only two cases occurred under two years of age. One of the chief causes was over-crowding among the poorer classes. Contagion is usually accidental, rarely venereal, according to Comby. Many women, incapable of transmitting gonorrhœa to their husbands, have vaginal discharges which are contagious to children. Contagion, then, takes place from one child to another, directly or indirectly. Acute contagious vulvo-vaginitis must be distinguished from aphthous and impetiginous vulvitis, from anæmic leucorrhœa, and from traumatic or irritative vulvitis due to onanism, oxyurides, etc. All these are non-specific and easily curable. Most cases of vulvo-vaginitis are gonococcal. In eighty-three cases observed by Spaulding, gonococci were found in fifty-six. Relapses are common, apart from reinfection; hence treatment should be continued some time after the discharge has ceased. The duration of this affection is indefinite and may be several

years. Complications include urethritis, stomatitis, cystitis, proctitis, purulent ophthalmia, arthritis, and peritonitis. According to Comby gonococcal peritonitis in little girls is not grave, and he mentions eight cases which recovered without operation. Epstein and others, however, have reported cases which were fatal, with or without laparotomy. Other observers have reported purulent salpingitis, and some think that vulvo-vaginitis may lead to malformations of the uterus, dysmenorrhœa, or even sterility, and that certain cases of suppurative salpingitis of doubtful origin in virgins may be due to gonococcal infection dating from infancy. As regards treatment, Comby reports favourable results from vaccines, the vaginal discharge having ceased in ten cases after two to four injections made every three or four days.

C. F. MARSHALL.

The treatment of gonococcus vulvo-vaginitis, with further observations on the value of the complement fixation test in the management of this disease (*Am. Journ. Dis. Child.*, 1914, VII, p. 230).—**Gilbert Smith** takes a more optimistic view of the curability of vulvo-vaginitis, and, from the evidence of the complement fixation test, concludes that the majority of cases are cured by efficient treatment. A negative reaction indicates a cure, but a weakly positive reaction does not indicate that the disease is still present, for it may be due to a "group reaction," in which the gonococcus antigen combines with antibodies of allied micrococci. As regards local treatment the author recommends vaginal irrigation with warm solutions of boric acid, sodium bicarbonate, or permanganate of potassium (1 in 8000 to 1 in 6000), followed by injection of about half a drachm of argyrol 10–20 per cent., or protargol 5 per cent., twice daily. In chronic cases irrigation with 1 in 1000 silver nitrate and application of tincture of iodine to the cervix through an endoscope tube. For urethritis he uses injection of about half a drachm of 5 per cent. argyrol. He considers that vaccines are of little use in gonorrhœa of mucous membranes. Local treatment should be continued for a month after symptoms have subsided.

C. F. MARSHALL.

Diabetes in early infancy (*Johns Hopkins Hosp. Bull.*, 1913, XXIV, p. 274).—**J. H. M. Knox (Jun.)** says that out of 6496 deaths occurring among diabetics only eight were under one year. A case is then quoted of diabetes in a breast-fed infant girl of healthy parentage, aged 9 months. The child was passing urine with 4 to 8 per cent. of glucose shortly after admission. Acetone became greatly increased, and within a week unconsciousness developed, and death took place eleven days after admission. At the autopsy there were patches of broncho-pneumonia in both lungs. Macroscopically there were no other very marked changes. Microscopically the islands of Langerhans in the pancreas were diminished in size and number, and the thyroid showed some evidence of increased functional activity by the misshapen acini. The case was characterised by insidious onset but rapid progress, and symptoms only existing for three weeks, rapid loss of weight amounting to 2 lb. in five days, and the absence of greatly increased thirst or hunger. The author then analyses sixteen cases in detail. He finds the disease, though rare in infancy, is becoming more frequent; the sexes are equally affected in infancy, while in after-life males show a striking preponderance. In three the disease was recognised a few days after birth, in thirteen several months elapsed before a diagnosis was made. In two accidents were assigned as the cause, in six injury to the central nervous

system was the ætiological factor, and in two injury during delivery may have been a predisposing circumstance. In a few instances, diabetes followed the daily ingestion of huge amounts of sugar. The chief symptoms in the sixteen cases were first restlessness, followed by increased thirst, hunger, and the passage of a large amount of urine. Loss of weight was a marked symptom. Three cases ended in coma. Thirteen of the sixteen died, and three apparently recovered, but the first two of the latter were mild. The severer cases died in three weeks, one survived for two years, and others survived several months. Urinary findings are naturally incomplete. In only one of the sixteen was the amount accurately measured, and here 680 c.c. were passed in twenty-four hours. The sugar varied in quantity from 1 to 10 per cent.; albumin was never present in any considerable amount. Attention was often directed to the condition by the stiffness of the napkins after drying and in one by the swarms of flies. The specific gravity varied from 1010 to 1036. Glycosuria occurs in gastro-intestinal disorders and in eczema. One of the sixteen was successfully treated on a diet consisting of the curd from a litre of milk and 100 grm. of cream suspended in 900 grm. of oatmeal. The cream was gradually increased to 300 grm. when sugar reappeared in the urine to the amount of 4 to 8 per cent. in 600 or 700 c.c. of urine. Oatmeal gruel, a little bouillon, and a green vegetable were then added to the milk mixture, with the result that the sugar tolerance was increased up to 16 grm. a day. On this diet there was no loss of body-weight, and at the close of the report the infant was doing well.

CHRISTOPHER ROLLESTON.

Three cases of diabetes mellitus in one family (*Arch. of Ped.*, 1914, xxxi, p. 764).—**C. G. Moore**.—The father, aged 40 years, a son, aged 19 years, and a daughter, aged 7 years, were affected within five months. In all the illness started suddenly with fever. The son died of coma within three months. The author tried autotherapy with the girl, as he had known cases of icterus irresponsive to all medicinal treatment clear up very soon when they were given their own urine to drink. The girl was therefore given 3 oz. of her own urine thrice daily, the colour and taste being masked. The percentage of sugar dropped, and there was some improvement in the general condition, but the treatment was not continued, and the child got worse. The issue is not recorded.

J. D. ROLLESTON.

Death during lumbar puncture (*La Pediatria*, 1914, xxii, p. 286).—**F. Fonzo** records a case of cerebro-spinal meningitis in a child, aged 8 months, where death occurred from paralysis of the respiratory centres during lumbar puncture. The pulse remained almost normal during the syncopal attacks, while the respiration became progressively feeble till it stopped. The author thinks that in order to facilitate the entrance of the trochar the child was held with its head strongly flexed forward and that the sudden movement thus made, given the existing equilibrium of pressure between the spinal space and the cerebral space, by the failure of the communication between the ventricular fluid and the sub-arachnoid spaces of the spinal cord, there was a displacement of the liquid mass forwards, thus causing a hyperæmia *ex vacuo* in the bulbar region.

VINCENT DICKINSON.

Typhoid cerebro-spinal meningitis in a newborn infant (*Le Nourrisson*, 1914, II, p. 211).—**M. Garnier** and **Havelacque**.—A male infant, aged 3 weeks, presented meningeal symptoms, and lumbar puncture

performed two days after the onset showed the presence of purulent meningitis. The disease ran a slow course characterised by moderate and irregular fever, repeated convulsions, disturbance of respiratory rhythm, and towards the end by generalised rigidity and opisthotonos. A week before death hæmatemesis and melæna occurred. Death took place on the fortieth day. The spinal meningitis was then cured, lumbar puncture giving issue to a normal fluid before death. Post mortem the spinal meninges and cord showed no lesion, the inflammation being confined to the brain, especially the ventricles, which contained pus. There was no trace of old or recent ulceration in the intestines. Bacteriological examination of the cerebro-spinal fluid showed typhoid bacilli. The source of infection could not be discovered, as the child had been exclusively breast-fed by the mother, who had not had typhoid fever.

J. D. ROLLESTON.

Pneumococcic meningitis and meningismus ('*Arch. of Ped.*,' 1914, xxxi, p. 323).—**F. Huber** records four cases of pneumonia in children, aged from 1 to 11 years, who showed cerebral symptoms with coma. The cerebro-spinal fluid was clear, under high tension, and sterile. All recovered. Huber regards the so-called "meningismus" as due to meningitis or encephalitis, which may only be visible microscopically and not be found on naked eye examination. Cases of meningismus which recover may subsequently develop mental defects.

J. D. ROLLESTON.

Acute Malta fever meningitis ('*Policlínica*,' 1914, II, p. 813).—**J. A. Jordán**.—A boy, aged $2\frac{1}{2}$ years, developed a form of an irregular type accompanied by slight and transient arthralgia. As he was living in an intensely malarial district he was subjected to an energetic treatment with quinine, but without effect. A month after the onset of the disease an agglutination test and blood culture were made and proved positive for the *Micrococcus melitensis*, and completely negative for typhoid and paratyphoid bacilli, *B. coli*, and the malarial parasite. An autogenous vaccine was used containing 5, 10, 15, 20 and 25 millions to one cubic centimetre, but no improvement resulted, and signs of meningitis developed. Lumbar puncture gave issue to clear fluid under hypertension showing numerous *Micrococci melitenses*. Intraspinal injection of anti-Malta fever serum was unsuccessful, and death occurred three weeks after the onset of the disease.

J. D. ROLLESTON.

Incomplete cerebro-spinal meningitis ('*Monde Méd.*,' 1914, xxiv, p. 129).—**A. Netter**, as a result of his observations, believes—(1) That the classic and characteristic symptoms of meningitis are often transitory and only noticed by the patient's friends. (2) When any sign is present at any time, especially if persistent fever exists with oscillations, lumbar puncture should be performed. (3) The fluid collected is not always purulent or sero-purulent, but may be clear, and should always be examined by the microscope.

F. R. B. ATKINSON.

Refractory or so-called "fast" cases of meningococcus meningitis ('*Am. Journ. Dis. Child.*,' 1914, viii, p. 307).—**H. Heiman** records two cases in children, aged 6 and $7\frac{1}{2}$ years. Both came from the same neighbourhood, were infected at about the same time, and failed to react to repeated injections of antimeningitis serum. Recovery eventually took place, in one fifty-eight days after admission to hospital, and in the other after an illness of nearly sixteen weeks.

J. D. ROLLESTON.

Influenzal meningitis (*Med. Journ. of Austral.*, 1914, I, p. 415).—**A. H. Tebbutt** describes a fatal case in a child, aged 8 months. The organism found resembled the Pfeiffer's bacillus. F. R. B. ATKINSON.

A fatal case of meningitis associated with an influenza-like bacillus (*Med. Journ. of Austral.*, 1914, I, p. 421).—**E. Atkinson** describes this case occurring in a child, aged 3 years and 10 months.

F. R. B. ATKINSON.

Influenzal meningitis (*Am. Journ. Obst.*, 1913, LXVII, p. 1031).—**T. S. D. Grasty**.—Pfohl in 1892 reported three cases of meningitis due to the Pfeiffer's bacillus. In 1903 Mya reported that this organism produced a rapidly fatal meningitis, either by a general infection or by the sinus being caused from a diseased joint, naso-pharynx, lung, or ear. Infants are affected in 60 per cent. of the cases and males more frequently than females. The average duration varies from five to twenty-six days. The lesions found post mortem resemble those of pneumococcal or meningococcal infection. The cerebro-spinal fluid is in most cases turbid and its lesion increased. The position of the cerebral lesions varies with the point of entry. In the cord the lesions follow the course of the vessels. In influenzal meningitis the leucocyte count is low, rarely exceeding 15,000, in marked contrast to other infections, where the numbers vary from 25,000 to 40,000. Kernig's and Babinski's signs are present, but the diagnosis can only be made by lumbar puncture and the subsequent recovery in pure culture of the bacillus. The following are possible complications: encephalitis, optic neuritis, epidural abscess, myelitis, poliomyelitis, and hemiplegia. The prognosis is grave, the mortality being about 85 per cent.

CHRISTOPHER ROLLESTON.

Tuberculous meningitis complicated by influenzal meningitis (*Am. Journ. Dis. Child.*, 1914, VIII, p. 150).—**H. K. Faber** records a case in a male infant, aged 5 months, admitted to hospital for convulsions. The cerebro-spinal fluid was clear, under normal pressure, and showed 145 cells per c.mm., 92 per cent. of which were lymphocytes. No organisms were found in the smear. A second lumbar puncture made next day showed a similar fluid, but a smear showed a number of organisms morphologically identical with *B. influenzae*, and a blood culture taken at the same time showed a profuse growth of *Staphylococcus albus* and *B. influenzae*. Three days later a choroid tubercle was found in the left fundus, and tubercle bacilli were found in the cerebro-spinal fluid. The symptoms differed from those of uncomplicated tuberculous meningitis by the almost continual clonic convulsions, and a long continued intermittent type of temperature. Death took place sixteen days after admission. The necropsy showed glandular pulmonary and generalised tuberculosis. In the brain there were two distinct processes: (1) The pia was everywhere studded with miliary tubercles, and there was a fibrino-purulent exudate at the base. (2) An abundant greyish-yellow purulent exudate over the convexity of the left hemisphere, a smear from which showed *B. influenzae* only. Reference is made to Hemenway's case (*vide BRITISH JOURNAL OF CHILDREN'S DISEASES*, 1908, V, p. 39), in which the spinal fluid was clear before death, but post mortem a purulent exudate on the parietal lobe was found, from which the pneumococcus was cultivated.

J. D. ROLLESTON.

Meningococcal meningitis and Koch's bacilli (*Arch de Méd. des Enf.*, 1914, XVII, p. 591).—**Loubet, Auban, and Riser**.—A boy, aged

9 years, who had had an anal fistula for the last five years, developed symptoms of cerebro-spinal meningitis. The cerebro-spinal fluid was turbid, and showed an excess of polymorphs and meningococci. The presence of a fistula prompted a search for tubercle bacilli, which were found in the blood and in the cerebro-spinal fluid after homogenisation. In the last lumbar puncture pneumococci were also present. No autopsy.

J. D. ROLLESTON.

Tuberculous and meningococcal or parameningococcal meningitis in a child (*Gaz. hebd. des Sci. méd. de Bordeaux*, 1914, xxxv, p. 135).—**E. Dubourg**.—A boy, aged 8 years, who had been suffering from tracheo-bronchial adenopathy, gradually developed symptoms of meningitis. Lumbar puncture gave issue to a clear fluid containing 90·21 per cent. lymphocytes and 8·83 per cent. polynuclear neutrophils, and showing the co-existence of tubercle bacilli and a diplococcus not staining by Gram. The child left the hospital and died shortly afterwards, before the identity of the cocci could be determined by another lumbar puncture.

J. D. ROLLESTON.

Internal pachymeningitis in young children (*New York Med. Journ.*, 1914, xcix, p. 720).—**A. Gordon**.—This may be traumatic or due to other causes such as syphilis, tuberculosis, rickets, or scurvy, and occasionally acute specifics. Some say hæmorrhage occurs first and inflammation afterwards, but probably the inflammation is the primary lesion. It occurs chiefly over the convexity of the brain. It develops insidiously with hydrocephalus, increased reflexes, spasticity of the limbs and sometimes convulsions, strabismus, vomiting, and high tension of the cerebro-spinal fluid. There may be retinal hæmorrhages. Gordon relates a case in a male child, aged 13 months, who had convulsions when six months old and again at 9 months. The child was semiconscious, with spastic arms and legs and increased reflexes, internal strabismus, and marked retinal hæmorrhages. Temperature 101° to 105° F., oscillating daily. Wassermann's reaction was negative in the blood serum and cerebro-spinal fluid; the latter was under high pressure and showed slight lymphocytosis but no organisms. Craniotomy was performed; a large cyst filled with cerebro-spinal fluid was found over the right motor area and emptied. The operation only relieved the symptoms for three weeks, they then returned and in spite of repeated lumbar punctures the child died six weeks later. Post mortem, a blood-clot was found on the inner surface of the dura. The second case was in a boy, aged 5 months, who for a year had similar symptoms to the other case. The cerebro-spinal fluid was negative but under great pressure. The boy died from convulsions. The autopsy showed thickened dura mater with much excess of serous fluid. Gordon considers the state of the cerebro-spinal fluid an important element in the diagnosis.

J. PORTER PARKINSON.

A hitherto undescribed form of chronic meningitis in children (*La Pediatria*, 1914, xxii, p. 181).—**A. Jovane** gives details of three cases. The ætiology is obscure; the presence of tobacco, lead, alcohol, tubercle, syphilis, and malaria, as well as hereditary neuropathy, being negatived. The disease commences with tremor in one limb, after twenty-four to forty-eight hours spreading to the others, the head, tongue, and trunk. In the first few days the temperature is raised, but this often escaped the notice of the parents. There is no interference with functions of any organs, no vomiting or convulsions. The essence of the disease is tremor with mus-

cular hypertonies, followed by contractures of varying degree. The tremor is characterised by irregular oscillations, usually frequent and fine, 120 to 160 a minute. It is accentuated by intentional movement, in crying and other emotional states, diminishes and sometimes disappears during sleep. Owing to the association of the tremor and hypertonus, the patient assumes a characteristic physiognomy, especially when the motor disturbance invades the whole body. The head is in continuous oscillatory movement and more or less dragged backwards from the retraction of the nuchal muscles. The buccal aperture is usually open, and through it can be seen the oscillating tongue. The voice is quavering and bleating like a goat's. The trunk in less marked cases is little affected, but in severe forms presents a curve backwards, a true opisthotonos which is sometimes lateral according as the spinal muscles are equally contracted or the contrary. The tremor, however, is communicated to the whole of the trunk. The upper extremities have a characteristic attitude. The arm is drawn away from the trunk, the forearm is flexed on the arm so that the back of the wrist rests on the chest and is rotated outward. The hand itself is prone, the thumb pressed on to the index finger, which is usually in semi-flexion, while the last three fingers are strongly flexed on the palm. The lower limbs are nearly always flexed, the feet in equinus position, the toes in plantar flexion. There is considerable resistance in the adductors. The tremor in the lower limbs is much less than in the upper. The reflexes, superficial and deep, are all exaggerated. Clonus and the signs of Troussseau and Babinski are absent. The electrical excitability of the nerves is increased. Lumbar puncture gives a very clear liquid under increased pressure; the centrifugalised deposit is scanty and contains a few leucocytes with occasional prevalence of polynuclears. The cases usually recover after many months or even a year. Treatment consists in baths daily at a temperature of 34° C., iodine internally, and repeated lumbar puncture. This paper, which was communicated to the Congresso Pediatrico at Bologna in 1913, well repays perusal.

VINCENT DICKINSON.

Reviews.

THE HEART IN EARLY LIFE. By G. A. SUTHERLAND, M.D., F.R.C.P.
Oxford Medical Publications. London: Henry Frowde and Hodder
and Stoughton. 1914. Price 6s. net.

If all medical text-books were as easy and pleasant to read as this one, publishers would have no use for their customary appeals to "the busy practitioner" to examine their wares. In a small space, Dr. Sutherland has given us a vast deal of information, but so skilfully has he besprinkled his text with examples and with aphorisms that the reader performs the task of mental digestion without conscious effort. After a brief introduction he gives an account of the functional cardiac disturbances of childhood, laying especial stress on the "sinus" type of arrhythmia and the importance of the respiratory movements as a factor in its production. He also has much to say about the inorganic murmurs of childhood. In both of these subjects he insists on the fallacy of regarding as important a single symptom unsupported by other evidences of organic heart disease, remarking that a "murmur should not be treated like a hot-house plant." After a brief

section devoted to paroxysmal tachycardia—a borderland between functional disturbance and organic disease—he gives an account of the organic cardiac lesions encountered in pædiatric practice. Throughout this section he insists on the paramount importance of rheumatic infection as a cause of the acquired lesions. He takes the broader view of this infection, accepting the type of disease encountered as strong evidence of its rheumatic origin, even in the absence of a history of rheumatism or chorea. He emphasises the fact that all parts of the heart are usually injured by the infection, and that the essential function of the heart is that of muscular contraction. The importance of rheumatic lesions, according to this teaching, lies in the degree to which they cripple the muscular functions, and particularly in respect to the left ventricle. In regard to treatment he gives much practical advice, such as should commend the book to many readers. It is a relief to find that so cautious and experienced an observer retains some confidence in the value of the salicylates in acute rheumatic carditis. Congenital malformations are not dealt with systematically, and this is the only criticism we have to offer, that his readers would be glad if Dr. Sutherland, in future editions, could give them the benefit of his experience in this direction also. A word must be added about Dr. Sutherland's attitude towards the newer graphic methods. He is enthusiastic as to their value and interest, but without being hypnotised into regarding them as anything more than means to an end. Throughout his book he insists on the importance of looking at heart disease from every point of view, and gaining thereby such accurate views as alone can lead to successful diagnosis, prognosis, and treatment. In conclusion, the book avoids technicalities, and no one interested in clinical medicine will find it hard to read.

C. C.

ZUR PROGNOSE UND ÄTIOLOGIE DER KINDER-HYSTERIE. Von Dr. A. TOBIAS. Berlin: S. Karger, 1913. Price 3·50m.

DR. TOBIAS has set herself to find out what was the subsequent history of the children under fourteen who had passed through Feer's clinic in Heidelberg with the diagnosis of hysteria during the years 1885-1903. After excluding several cases for various grounds there were left thirty for investigation. She was able to see nineteen of the former patients and for the rest had to be content with replies to written questions, but of the thirty there were twenty-one cases of pure hysteria; the others were epileptics or hysteria complicating various physical disorders. A short account of each case and the later history is given, but of the twenty-one cases Tobias claims that sixteen have been permanently cured. Admitting with the author that the figures are too small for any sweeping judgment, and further that the histories, whether obtained in writing or in person, are somewhat unreliable, the results are certainly more hopeful than are usually entertained with the hysteria of children. On the other hand, although the subsequent histories have been obtained after menstruation in the girls, most of the former patients were still in adolescence or, at all events, had not undertaken any position of independence or responsibility, *e. g.* of the girls only one is married. Nevertheless, the writer justifies her conclusion that the French school is unnecessarily pessimistic. Under favourable conditions a large number of cases of hysteria in childhood, even with a recognisable psychopathic basis can be permanently cured.

M. D. E.